Exposure Assessment of Trichloroethylene

Chieh Wu and John Schaum

Office of Research and Development, National Center for Environmental Assessment, U.S. Environmental Protection Agency, Washington, DC USA

This article reviews exposure information available for trichloroethylene (TCE) and assesses the magnitude of human exposure. The primary sources releasing TCE into the environment are metal cleaning and degreasing operations. Releases occur into all media but mostly into the air due to its volatility. It is also moderately soluble in water and can leach from soils into groundwater. TCE has commonly been found in ambient air, surface water, and groundwaters. The 1998 air levels in $\mu g/m^3$ across 115 monitors can be summarized as follows: range = 0.01–3.9, mean = 0.88. A California survey of large water utilities in 1984 found a median concentration of 3.0 µg/L. General population exposure to TCE occurs primarily by inhalation and water ingestion. Typical average daily intakes have been estimated as 11–33 µg/day for inhalation and 2–20 µg/day for ingestion. A small portion of the population is expected to have elevated exposures as a result of one or more of these pathways: inhalation exposures to workers involved in degreasing operations, ingestion and inhalation exposures occurring in homes with private wells located near disposal/contamination sites, and inhalation exposures to consumers using TCE products in areas of poor ventilation. More current and more extensive data on TCE levels in indoor air, water, and soil are needed to better characterize the distribution of background exposures in the general population and elevated exposures in special subpopulations. *Key words*: exposure assessment, TCE, trichloroethylene. — *Environ Health Perspect* 108(suppl 2):359–363 (2000).
http://ehpnet1.niehs.nih.gov/docs/2000/suppl-2/359-363wu/abstract.html

Purpose

This article reviews exposure information available for trichloroethylene (TCE) and assesses the magnitude of human exposure. Background information is provided on TCE sources, environmental fate, and levels in the environment and people. The results include findings on exposure pathways, potentially exposed populations, range of estimated exposures, and associated uncertainties.

Background

Sources

Releases of TCE into the environment occur during its manufacture, use, and disposal. The major use of TCE is as a degreaser for metal cleaning operations. It is also used as a paint stripper, adhesive solvent, ingredient in paints and varnishes, and in the manufacture of organic chemicals. Releases from nonanthropogenic activities are negligible, although TCE can be created by one red microalga and in temperate, subtropical, and tropical algae (*1*).

Most TCE used in the United States is released to the atmosphere from vapor degreasing operations (*2*). Releases to air also occur at sewage treatment and disposal facilities, water treatment facilities, and landfills (*2*). TCE has been detected in stack emissions from municipal and hazardous waste incinerators and it could be released to surface waters from industrial discharges of wastewater streams as well as to groundwater via leaching from landfills (*2*).

Table 1 reports the releases of TCE for years 1987–1994 on the basis of the U.S. Environmental Protections Agency's (U.S. EPA) Toxic Release Inventory (*3*). The releases have ranged from 55.6 million pounds in 1987 down to 29.9 million pounds in 1994. The table shows that TCE releases to the air dominate over other types and that total releases have generally declined since 1987.

Environmental Fate

In terrestrial environments, the dominant fate of TCE released to surface soils is volatilization to the air. Because of its moderate water solubility, TCE introduced into soil (e.g., landfills) has the potential to migrate through the soil into groundwater. The relatively frequent detection of TCE in groundwater confirms its mobility in soils. Biodegradation in soil and groundwater is thought to be slow (half-life on the order of months to years) (*4*).

Because of its high vapor pressure, TCE in the atmosphere is expected to be present primarily in the vapor phase rather than sorbed to particulates. Some removal by scavenging during wet precipitation is expected because of the moderate solubility of TCE in water (1.1 g/L). The major degradation process affecting vapor-phase TCE is photooxidation by hydroxyl radicals (half-life on the order of 1–11 days) (*5*).

The dominant fate of TCE released to surface waters is volatilization (predicted half-life of minutes to hours). Bioconcentration, biodegradation, and sorption to sediments and suspended solids are not thought to be significant (*5*).

Exposure Media Concentrations

TCE has been detected in the air throughout the United States. The ambient air measurement data for TCE were obtained from the Aerometric Information Retrieval System (AIRS) using the AIRS website: *http://www.epa.gov/airsdata* (*6*). These data were collected from a variety of sources including state and local environmental agencies and cover the years 1985–1998. They represent about 1,200 measurements from 25 states. The most recent data (1998) come from 115 monitors located in 14 states. The 1998 air levels in $\mu g/m^3$ across all 115 monitors can be summarized as follows: range = 0.01–3.9; mean = 0.88, 50th percentile = 0.32 and 90th percentile = 1.76. Table 2 summarizes the data by year, showing the average and number of samples. Relatively few samples were collected in 1985 and 1986, but each year after 1986 is represented by at least 50 samples. The data suggest a general downward trend from about 1.5 $\mu g/m^3$ in the late 1980s to 0.8 $\mu g/m^3$ in the late 1990s. Table 3 shows the monitoring data organized by land setting (rural, suburban, or urban) and land use (agricultural, commercial, forest, industrial, mobile, and residential). Urban air levels are about 3 times higher than rural areas. Among the land use categories, TCE levels are highest in commercial/industrial areas and lowest in forest areas.

TCE ambient air concentrations in 1990 were modeled for all census tracts of the continental United States as part of the U.S. EPA Cumulative Exposure Project (CEP, see *www.epa.gov/cumulativeexposure/air/air.htm*) (*7*). A variety of sources were used to obtain emissions data and the air modeling was done using a Gaussian dispersion model. Table 4 shows the distribution of modeled TCE

This article is part of the monograph on Trichloroethylene Toxicity.

Address correspondence to C. Wu, National Center for Environmental Assessment (8623 D), U.S. EPA ORD, Washington, DC 20460. Telephone: (202) 564-3257. Fax: (202) 565-0076. E-mail: wu.chieh@epa.gov

The authors thank Versar, Inc., for their U.S. EPA contract report "Sources, Emission and Exposure for Trichloroethylene (TCE)," which provides most of the background information used in this article. The authors also thank C. Scott and M. Dusetzina for their review and comments of the draft report.

The views expressed in this paper are those of the authors and do not necessarily reflect the views or policies of the U.S. Environmental Protection Agency.

Received 20 October 1999; accepted 23 February 2000.

Table 1. Annual releases of trichloroethylene in the United States (lb/year).

Year	Number of reporting facilities	Fugitive air releases	Stack air releases	Surface water releases	Underground injection	Land disposal	POTW transfer	Other transfers	Total
1987	959	25,978,879	29,436,952	30,104	18,720	56,733	130,178	11,689,590	67,341,156
1988	951	26,168,126	29,759,510	13,801	390	21,186	85,652	6,509,867	62,558,532
1989	899	22,629,351	27,054,328	15,849	390	8,686	31,519	4,962,054	54,702,177
1990	807	19,030,377	20,900,640	14,285	805	12,554	11,949	3,879,599	43,850,209
1991	724	17,078,485	18,860,997	12,784	800	62,991	73,195	10,625,967	46,715,219
1992	681	15,585,757	14,866,100	8,606	466	20,726	70,149	9,807,719	40,359,523
1993	790	14,524,316	15,939,964	5,220	460	8,212	42,987	10,143,591	40,664,750
1994	783	14,788,788	15,083,085	1,671	288	4,417	50,325	12,307,585	42,236,159

POTW, public-owned transport works. Data from U.S. EPA (*3*).

Table 2. Mean TCE air levels across monitors by year.[a]

	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	1995	1996	1997	1998
Mean concentration (μg/m^3)	1.4	1.39	1.68	4.87	1.69	1.84	2.86	1.37	1.12	0.95	0.78	0.65	0.74	0.88
n	11	21	53	57	96	59	70	76	84	89	146	150	129	115

[a]1 ppb = 5.36 μg/m^3.

Table 3. Mean TCE air levels across monitors by land setting and use (1985–1998).[a]

	Rural	Suburban	Urban	Agricultural	Commercial	Forest	Industrial	Mobile	Residential
Mean concentration (μg/m^3)	0.42	1.26	1.61	1.08	1.84	0.1	1.54	1.5	0.89
n	93	500	558	31	430	17	186	39	450

[a]1 ppb = 5.36 μg/m^3.

Table 4. Modeled TCE air concentrations in continental United States for 1990.[a]

	25th Percentile	50th Percentile	75th Percentile	95th Percentile	Maximum	Overall mean	Urban mean
Concentration (μg/m^3)	0.13	0.24	0.45	1.1	32	0.37	0.5

[a]1 ppb = 5.36 μg/m^3.

ambient air concentrations across the continental United States. The modeling suggests that 97% of the census tracts have TCE concentrations ranging from 0 to 1.5 μg/m^3. The average level was estimated as 0.37 μg/m^3 and the maximum as 32 μg/m^3. The averages and percentiles are better interpreted as population-weighted values than spatial averages because all census tracts have roughly equal populations but are more variable in geographic area. Figure 1 is a map of the CEP-modeled TCE air concentrations in New Jersey. The average across all population tracts in the state is 0.5 μg/m^3. The map indicates, however, that the vast majority of the state, on an area basis, has levels under 0.5 μg/m^3. Relatively high levels (generally 1–12 μg/m^3) were estimated for the densely populated areas around Camden and Newark–Paterson. The highest levels (up to 30 μg/m^3) were estimated for a few (presumably industrial) sectors within these areas. The CEP data suggest that this pattern (i.e., generally low TCE levels in rural areas, moderate levels in urban areas, and highest levels in small commercial/industrial sectors) is common across most states. The monitoring data, as discussed earlier, also suggest that this is the general pattern across the country.

These modeled values should be interpreted with caution. Clearly they are not as reliable as measured values for specific locations. As discussed earlier, the AIRS data show an average for 1990 across 59 monitoring stations of 1.84 μg/m^3. This is much higher than the national average from CEP of 0.37 μg/m^3. An important difference, though, is that the CEP estimate represents all areas of the continental United States, whereas the 1990 AIRS data for TCE represent only 59 monitors located in eight states. CEP compared modeled estimates with measured values in the same locations and found that for most chemicals, agreement was usually within a factor of 3, with underestimates being more common than overestimates. More variability, however, was found in the model–monitor comparisons for TCE than for other HAPs (hazardous air pollutants). In addition, the tendency for underestimation observed for other HAPs was not seen for TCE. The TCE model–monitor comparisons can be summarized as follows: model–monitor comparisons were made at 57 monitoring sites; the median of the model–monitor ratios was 0.76; arithmetic mean ratio = 2.33; geometric mean ratio = 1.02; 53% of ratios were less than 1.0; 51% were within a factor of 3 (i.e., within the range of 0.33–3.0); 19% were less than 0.33; and 30% were greater than 3.0.

TCE has been measured in rain, surface waters, groundwater, drinking water, and sea water (Table 5). According to the International Agency for Research on Cancer (IARC) (*1*), the reported median concentrations of TCE during 1983–1984 were 0.5 μg/L in industrial effluents and 0.1 μg/L in surface water. Results from an analysis of the U.S. EPA STORET database (*8*) (1980–1982) showed that TCE was detected in 28% of 9,295 surface water reporting stations nationwide (*2*). The Agency for Toxic Substances and Disease Registry (ATSDR) (*2*) has indicated that TCE is the most frequently reported organic contaminant in groundwater and has estimated that between 9 and 34% of the drinking water supplies tested in the United States may have some TCE contamination (*2*). The U.S. EPA Office of Groundwater and Drinking Water reported that most water supplies are in compliance with the maximum contaminant level (maximum contaminant level [MCL], 5 μg/L) and that only 407 samples out of many thousands taken from community and other water supplies throughout the country over the past 11 years (1987–1997) have exceeded the MCL limit for TCE (*9*).

TCE concentrations in groundwater have been measured extensively in California. The data were derived from a survey of large water utilities (i.e., utilities with more than 200 service connections). The survey was conducted by the California Department of Health Services (*10*). From January 1984 through December 1985, wells in 819 water systems were sampled for organic chemical contamination. The water systems use a total of 5,550 wells, 2,947 of which were sampled. TCE was found in 187 wells at concentrations up to 440 μg/L, with a median concentration of 3.0 μg/L. Generally, the most contaminated wells and the wells with the highest concentrations were found in the heavily urbanized areas of the state. Los Angeles County registered the greatest number of contaminated wells—149.

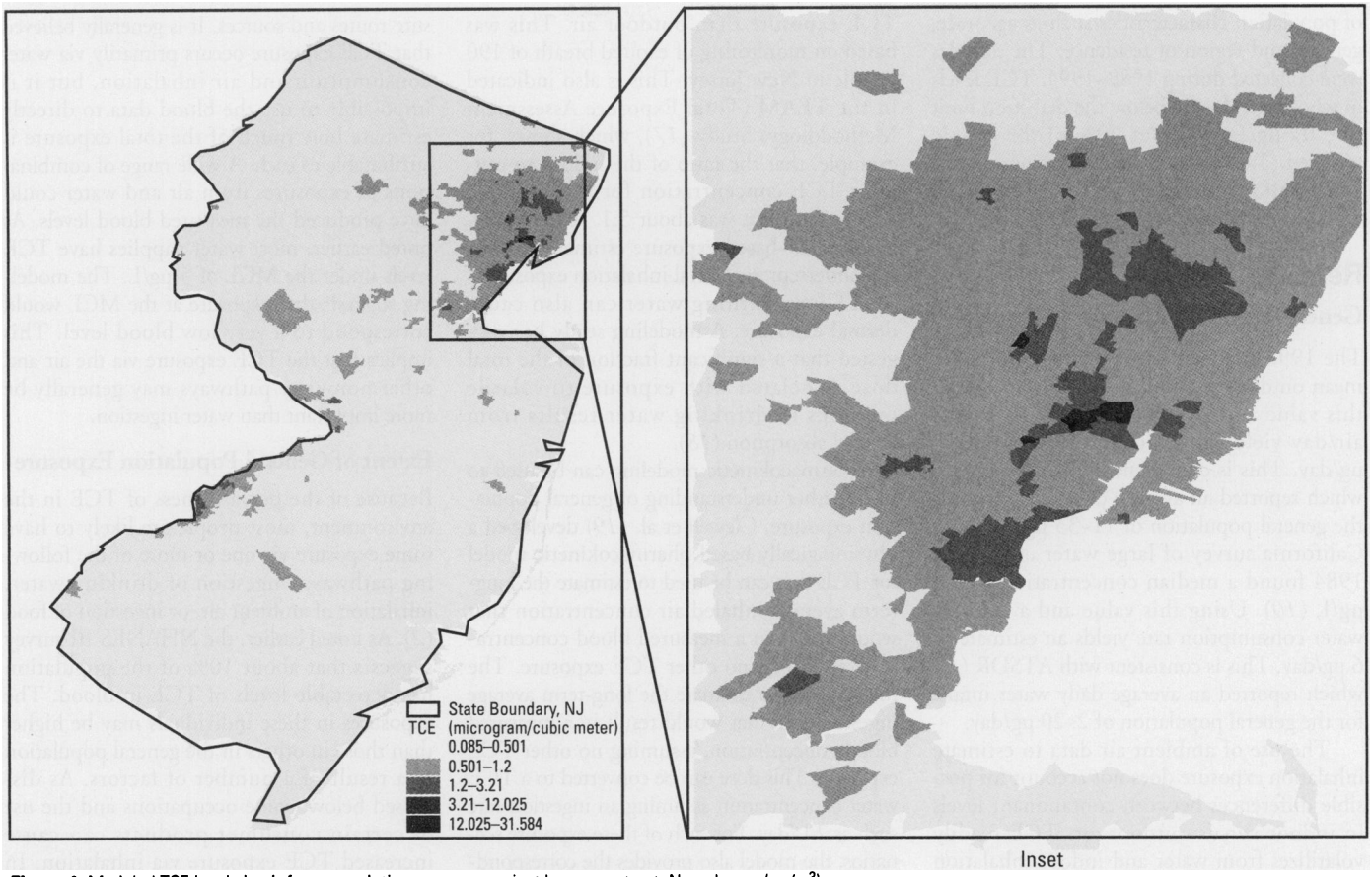

Figure 1. Modeled TCE levels in air from cumulative exposure project by census tract, New Jersey (μg/m^3).

TCE has been reported in marine sediments, marine invertebrates, marine mammals, foods, mother's milk, and human urine and blood (*1,5*). TCE was found in sediment and marine animal tissue collected in 1980–1981 near the discharge zone of a Los Angeles County waste treatment plant. Concentrations were 17 μg/L in the effluent, < 0.5 μg/kg in dry weight in sediment, and 0.3–7 μg/kg wet weight in various marine animal tissues (*1*). TCE also has been found in foods in the United States and the United Kingdom. Based on limited surveys, the TCE levels found in U.S. food were 0.9 μg/kg in grain-based foods, 1.8 g/kg in table-ready foods, 73.6 μg/kg in butter and margarine, 0.5 μg/kg in peanut butter, 3.0 μg/kg in ready-to-eat cereals, 1.3 μg/kg in highly processed foods, and 3.8 μg/kg in cheese products (*1*).

Table 5. TCE levels in water (μg/L).

Water type	Location	Year	Mean	Median	Range	Number of samples	Ref.
Industrial effluent	U.S.	83		0.5			(*1*)
Surface waters	U.S.	83		0.1			(*1*)
Rainwater	Portland, OR	84	0.006		0.002–0.02		(*25*)
Groundwater	MN	83			0.2–144		(*26*)
	NJ	76			< 1,530		(*27*)
	NY	80			< 3,800		(*27*)
	PA	80			< 27,300		(*27*)
	AZ				8.9–29		(*1*)
	MA	76			< 900		(*27*)
Drinking water	U.S.	76			0.2–49		(*1*)
	U.S	77			0–53		(*1*)
	U.S.	78			0.5–210		(*1*)
	NJ	84–85	23.4		Maximum 67	1,130	(*28*)
	CA	84			8–12	486	(*17*)
	CA	84	66			486	(*17*)
	NC	84	5			48	(*17*)
	ND	84	5			48	(*17*)
	MA				Maximum 267		(*1*)

Concentrations in Humans

Measurements of human body burdens provide a useful supplement to environmental monitoring for purposes of characterizing human exposure. Some environmental monitoring data, however, may not be representative of actual exposures. For example, ambient air monitors are fixed units typically located on top of buildings and do not directly sample the air that a person actually breathes throughout a day. Also, environmental monitoring does not represent all pathways by which exposure can occur, i.e., indoor air inhalation, smoking, food ingestion, etc. Biological monitoring studies have detected TCE in human blood and urine in the United States and other countries such as Croatia, China, Switzerland, and Germany (*1*). TCE has been most frequently detected in persons exposed through occupational degreasing operations (*1*). In 1982, TCE was detected in eight of eight human breast milk samples from four U.S. urban areas (*2,5*). The Third National Health and Nutrition Examination Survey (NHANES III) examined TCE concentrations in blood from 677 nonoccupationally exposed individuals (*11*). The individuals were selected to represent a range

of population characteristics such as age, race, gender, and region of residence. The samples were collected during 1988–1994. TCE levels in whole blood were below the detection limit of 0.01 μg/L for about 90% of the people sampled (Table 6). Assuming that nondetects equal half of the detection limit, the mean concentration was 0.017 μg/L (*11*).

Results

General Population Exposure Levels

The 1998 AIRS monitoring data indicate a mean outdoor air level of 0.88 μg/m^3. Using this value and an inhalation rate of 20 m^3 air/day yields an exposure estimate of 18 μg/day. This is consistent with ATSDR (*2*), which reported an average daily air intake for the general population of 11–33 μg/day. The California survey of large water utilities in 1984 found a median concentration of 3.0 μg/L (*10*). Using this value and a 2 L/day water consumption rate yields an estimate of 6 μg/day. This is consistent with ATSDR (*2*), which reported an average daily water intake for the general population of 2–20 μg/day.

The use of ambient air data to estimate inhalation exposure does not account for possible differences between contaminant levels in indoor versus outdoor air. TCE readily volatilizes from water and indoor inhalation exposure may be comparable to or greater than ingestion exposures in homes where the water supply contains TCE (*2,12–15*). For example, in two homes using well water with TCE levels averaging 22–128 μg/L, the TCE levels in bathroom air ranged from < 0.5 to 40 mg/m^3 when the shower was run less than 30 min (*13*). In one study, the transfer of TCE from shower water to air had a mean efficiency of 61% (independent of water temperature); it was concluded that a 10-min shower in TCE-contaminated water could result in a daily inhalation exposure comparable to that expected from drinking TCE-contaminated tap water (*2*). Indoor use of TCE-containing products can also contribute to exposures. Wallace et al. (*16*) concluded that indoor air contributes more to overall TCE exposure than outdoor air. This was based on monitoring of expired breath of 190 people in New Jersey. This is also indicated in the TEAM (Total Exposure Assessment Methodology) Study (*17*), which shows, for example, that the ratio of the indoor to outdoor TCE concentration for Greensboro, North Carolina, was about 5:1. Accordingly, ambient air-based exposure estimates probably underrepresent total inhalation exposures.

TCE in bathing water can also cause dermal exposure. A modeling study has suggested that a significant fraction of the total dose associated with exposure to volatile organics in drinking water results from dermal absorption (*18*).

Pharmacokinetic modeling can be used to gain further understanding of general population exposure. Clewell et al. (*19*) developed a physiologically based pharmacokinetic model for TCE that can be used to estimate the long-term average inhaled air concentration that would result in a measured blood concentration, assuming no other TCE exposure. The model can also estimate the long-term average ingested dose that would result in a measured blood concentration, assuming no other TCE exposure. This dose can be converted to a TCE water concentration assuming an ingestion rate such as 2 L/day. For each of these exposure scenarios, the model also provides the corresponding concentrations of trichloroacetic acid and dichloroacetic acid in blood and the amount of TCE metabolized per day. This model was applied to the range of TCE levels in blood as measured in NHANES III. Table 7 shows the resulting exposure estimates corresponding to the range of TCE blood levels. The TCE environmental concentrations modeled from blood levels exceeded the range of measured values for air and water: modeled mean concentration in drinking water was 59.5 μg/L (measured range was trace to 50 μg/L) and the modeled mean air concentration was 4.2 μg/m^3 (measured range was for 0.01–3.9 μg/m^3). This implies that neither inhalation nor water ingestion dominate exposure; rather both contribute to the total exposure. Exposure estimates derived from blood cannot distinguish among exposure routes and sources. It is generally believed that TCE exposure occurs primarily via water consumption and air inhalation, but it is impossible to use the blood data to directly estimate how much of the total exposure is attributable to each. A wide range of combinations of exposures from air and water could have produced the measured blood levels. As noted earlier, most water supplies have TCE levels under the MCL of 5 μg/L. The modeling suggests that exposure at the MCL would correspond to a very low blood level. This implies that the TCE exposure via the air and other nonwater pathways may generally be more important than water ingestion.

Table 6. TCE levels in whole blood by population percentile.[a]

	Percentile								
	10	20	30	40	50	60	70	80	90
Concentration (μg/L)	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.005	0.012

[a]Nondetects assumed equal to half the detection limit (0.01 μg/L). Data from IARC (*1*) and Ashley (*11*).

Table 7. Modeled exposure estimates for TCE.

	Air concentration (μg/m^3)	Ingested dose (μg/kg-day)	Water concentration (μg/L)
10th Percentile blood level (0.005 μg/L)	1.25	0.5	17.5
90th Percentile blood level (0.012 μg/L)	3.0	1.2	42.0
Mean blood level (0.017 μg/L)	4.3	1.7	59.5

Extent of General Population Exposure

Because of the pervasiveness of TCE in the environment, most people are likely to have some exposure via one or more of the following pathways: ingestion of drinking water, inhalation of ambient air, or ingestion of food (*2*). As noted earlier, the NHANES III survey suggests that about 10% of the population has detectable levels of TCE in blood. The exposures in these individuals may be higher than those in others in the general population as a result of a number of factors. As discussed below, some occupations and the use of certain consumer products can cause increased TCE exposure via inhalation. In addition, some members of the general population may have increased TCE exposure via their drinking water. The extent of TCE exposure via drinking water is difficult to estimate, but the following discussion provides some perspective on this issue.

TCE is the most frequently reported organic contaminant in groundwater (*2*); 93% of the public water systems in the United States obtain water from groundwater (*20*) and between 9 and 34% of the drinking water supply sources tested in the United States may have some TCE contamination (*2*). Although commonly detected in water supplies, the levels are generally low, since, as discussed earlier, MCL violations for TCE in public water supplies are relatively rare for any extended period (*9*). Private wells, however, are often not closely monitored and if located near TCE disposal/contamination sites where leaching occurs, may have undetected contamination levels. About 10% of Americans (27 million people) obtain water from sources other than public water systems, primarily private wells (*20*). TCE is a common contaminant at Superfund sites. It has been identified in at least 852 of the 1,416 hazardous waste sites (848 in the United States and 4 in Puerto Rico) proposed for inclusion on the U.S. EPA National Priorities List (NPL) (*2*). Studies have shown that many people live near these sites: 41 million people live less than 4 miles from one or more

of the nation's NPL sites, and on average 3,325 people live within 1 mile of any given NPL site (*21*). Thus, although exact estimates cannot be made, many people are probably exposed to TCE via drinking water from private wells. It is not known how often such exposures would be above the MCL.

Special Population Exposure

ATSDR (*2*) has reported that TCE exposures may be elevated for people living near waste facilities, residents of some urban or industrialized areas, and people exposed at work. As noted above, populations residing near hazardous waste sites might experience exposures to TCE higher than the average for the general population.

Occupational exposure to TCE in the United States has been identified in various degreasing operations, silk screening, taxidermy, and electronic cleaning (*1*). The National Institute for Occupational Safety and Health conducted a survey of various industries from 1981 to 1983 and estimated that approximately 401,000 U.S. employees in 23,225 plants are potentially exposed to TCE (*1,2*). Time-weighted average concentrations from personal monitoring ranged from 1.2 to 5.1 ppm (7,059–30,000 mg/m^3) at individual industrial sites where TCE was used (*2*).

Inhalation exposure may be elevated for individuals using products containing TCE in areas with poor ventilation (*2*). These products include wood stains, varnishes, finishes, lubricants, adhesives, typewriter correction fluids, paint removers, and cleaners (*2*). Use of TCE, however, has been discontinued in some consumer products (i.e., as an inhalation anesthetic, fumigant, and extractant for decaffeinating coffee) (*2*).

Because TCE has been detected in breast milk samples of the general population, infants who ingest breast milk may be exposed. Also, since TCE can be present in soil, children may be exposed through activities such as playing in or ingesting soil.

Discussion

The current data suggest that TCE exposure is widespread, but it is unclear how often the aggregate exposures across all pathways reach levels of concern. More current and more extensive data are needed to better characterize the full distribution of background exposures in the general population and elevated exposures in special subpopulations. Specifically more data are needed on the levels of TCE in private wells, indoor air, soil, food, blood across all ages, and mother's milk.

TCE metabolites and other parent compounds that produce the same metabolites can cause health effects similar to TCE. Ideally a complete risk assessment should consider the potential for additive impacts of these compounds. The primary metabolites of trichloroethylene include: dichlorovinylcysteine, trichlorovinylcysteine, chloral, chloral hydrate, trichloroacetic acid, dichloroacetic acid, and monochloroacetic acid. The primary parent compounds that produce the same metabolites as trichloroethylene include: tetrachloroethylene (PCE), methyl chloroform, 1,1,1,2-tetrachloroethane, *cis*-1,2-dichloroethylene, *trans*-1,2-dichloroethylene, 1,2-dichloroethylene, and 1,1-dichloroethane. The parent compounds are used in a wide variety of manufacturing industries as solvents, extractants, textile dry cleaners, metal degreasers, insulating fluids/coolants, and chemical intermediates. The metabolites have more restricted uses as chemical intermediates, herbicides, and pharmaceuticals. Relatively little information is available for most of these compounds in regard to environmental levels and exposure. The exceptions are PCE and 1,1,1-trichloroethane, which have been reasonably well studied. Like TCE, these two compounds are commonly found in air and water and cause exposure by inhalation and ingestion (*1,22–24*). Thus, background exposure to these related compounds may influence the effect of small incremental exposures of TCE. This issue should be considered in quantitative dose–response analyses.

References and Notes

1. IARC. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 63:Dry Cleaning, Some Chlorinated Solvents and Other Industrial Chemicals. Lyon:International Agency for Research on Cancer, 1995.
2. ATSDR. Toxicological Profile for Trichloroethylene. Update Draft for Public Comments. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1995.
3. U.S. EPA. Toxic Release Inventory. Washington, DC:U.S. Environmental Protection Agency, 1996.
4. Howard PH, Boethling RS, Jarvis WF, Meylan WM, Michalenko EM. Handbook of Environmental Degradation Rates. Chelsea, MI:Lewis Publishers, 1991.
5. Hazardous Substances Data Bank. National Library of Medicine. Washington, DC:National Institutes of Health, 1996.
6. U.S EPA. AIRSDATA. Available: *www.epa.gov/airsdata*. Research Triangle Park, NC:U.S. Environmental Protection Agency, 1999.
7. U.S. Environmental Protection Agency. Cumulative Exposure Project (CEP). U.S. EPA Internet. Available: *www.epa.gov/cumulativeexposure/air/air.htm* (1999).
8. U.S. EPA, STORET Database. Washington, DC:U.S. Environmental Protection Agency, 1982.
9. U.S. EPA. Data File. Washington, DC:U.S. Environmental Protection Agency, 1998.
10. DHS. Organic Chemical Contamination of Large Public Water Systems in California. Sacramento, CA:California Department of Health Services,1986.
11. Ashley DL, Bonin MA, Cardinali FL, McCraw JM, Wooten JV. Blood concentrations of volatile organic compounds in a non-occupationally exposed US population and in groups with suspected exposure. Clin Chem 40(7):1401–1404 (1994).
12. Andelman JB. Human exposure to volatile halogenated organic chemicals in indoor and outdoor air. Environ Health Perspect 62:313–318 (1985).
13. Giardino NJ, Gumerman E, Esmen NA, Andelman C, Wilkes J, Davidson CI, Small MJ. Shower volatilization exposures in homes using tap water contaminated with TCE. J Expo Anal Environ Epidemiol (suppl) 1:147–158 (1992).
14. Andelman JB, Meyers SM, Wilder LC. Volatilization of organic chemicals from indoor uses of water. In: Chemicals in the Environment (Lester JN, Perry R, Sterritt RM, eds). London:Selper Ltd., 1986.
15. Andelman J., Couch A, Thurston WW. Inhalation exposures in indoor air to trichloroethylene from shower water. Chap 16. In: Environmental Epidemiology (Kopfler FC, Craun GC, eds). Chelsea, MI:Lewis Publishers, 1986.
16. Wallace LA, Pellizzari ED, Hartwell TC. Personal exposures, Indoor-outdoor relationships and breath levels of toxic air pollutants measured for 355 persons in New Jersey. Atmos Environ 19:1651–1661 (1985).
17. U.S. EPA. The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis: Vol 1. EPA/600/6-87/002a. Washington, DC:U.S. Environmental Protection Agency, 1987.
18. Brown HS, Bishop DR, Rowan CA. The role of skin absorption as a route of exposure for volatile organic compounds in drinking water. Am J Public Health 74(5):479–484 (1984).
19. Clewell J, Gentry PR, Gearhart JM, Allen BC, Anderson ME. Considering pharmacokinetic and mechanistic information in cancer risk assessments for environmental contaminants: examples with vinyl chloride and trichloroethylene. Chemosphere 31(1):2561–2578 (1995).
20. U.S. EPA. The National Public Water Systems Supervision Program. The FY 1994 Compliance Report. Office of Water. EPA 812-R-94-001. Washington, DC:U.S. Environmental Protection Agency, 1995.
21. ATSDR. Biennial Report to Congress (1991 and 1992). Atlanta, GA:Agency for Toxic Substances and Disease Registry. Internet site: *www.dhhs.gov* (1996).
22. ATSDR. Toxicological Profile for *cis*-1,2-Dichloroethene, *trans*-1,2-Dichloroethene, 1,2-Dichloroethene. Update Draft. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1990.
23. ATSDR. Toxicological Profile for Tetrachloroethylene. Update Draft for Public Comments. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1995.
24. ATSDR. Toxicological Profile for 1,2-Dichloroethene. Update Draft for Public Comments. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1994.
25. Ligocki MP, Leuenberger C, Pankow JF. Trace organic compounds in rain. II: Gas scavenging of neutral organic compounds. Atmos Environ 19:1609–1617 (1985).
26. Sabel GV, Clark TP. Volatile organic compounds as indicators of municipal solid waste leachate contamination. Waste Manag Res 2:119–130 (1984).
27. Burmaster DE. The new pollution-groundwater contamination. Environment 24(2):6–36 (1982).
28. Cohn P, Klotz J, Bove F, Berkowitz M, Fagliano J. Drinking water contamination and the incidence of leukemia and non-Hodgkin's lymphoma. Environ Health Perspect 102:556–561 (1994).